Hypnogogic hallucinations occur more frequently in the general population than expected.

NON-PSYCHOTIC HALLUCINATIONS

DEAR EDITOR:

Hypnogogic hallucinations, so named by Alfred Maury and viewed as "psychosensory hallucinations" by Baillarger in the 1840s, have been reported consistently across time and cultures since the Renaissance Age.[1,2]

Hypnogogic sleep phenomena are defined as occurring upon awakening and experienced in the semi-conscious state. They often involve vivid auditory, visual, and tactile misperceptions or hallucinations. These hallucinations appear to represent the rapid transition from a sleep stage into a state of wakefulness.[3]

The prevalence of this phenomenon in the general population is undefined. A survey study of 870 university students found that 22.5% reported at least one hallucinoid sleep occurrence.[4] An investigation study by method of telephone sampling of the noninstitutionalized general population of the United Kingdom, Germany, and Italy (N=13,057), aged 15 years or over, found that 24.8 percent of respondents experienced hypnogogic hallucinations. Hypnogogic hallucinations were found to be much more common than expected, with a prevalence that far exceeded that which could be explained by the association with narcolepsy, estimated to be only 0.04 percent in the same populations.[5]

A review of neuroimaging and electrophysiological studies validate quantitative differences between the two apportioned phases of sleep: rapid eye movement (REM) and non-rapid eye movement (NREM).[6] Sleep begins with stage 1 of non-REM, which is characterized by low-amplitude, mixed frequency electroencephalographic (EEG) activity. Stage 2 is represented by the appearance of moderately low voltage EEG alpha-wave bursts of activity. Stage 3 is one of lower frequency and increased amplitude of EEG waves.

In stage 4, high-voltage delta waves at maximum slowing are found on EEG, which is characteristic of deep sleep and synchronization of slow waves.[7,8] Within NREM sleep, most central deactivated areas are located in the pons, mesencephalon, cerebellum, thalami, basal ganglia, basal forebrain-hypothalamus, prefrontal cortex, and in the temporal lobe.[9] During REM sleep, rapid, low-voltage, irregular EEG activations can be found in the pontine tegmentum, thalamic nuclei, limbic region, and in the temporo-occipital areas. In contrast, the dorso-lateral prefrontal cortex and the left parietal cortex are the least active brain regions.[9,10]

REM sleep-associated dreaming has been described as a form of hyperattentional processing mediated by a graduated increase in activity of cortical cholinergic inputs and their cortical interactions in the course of activated thalamic efferents.[10] Furthermore, during REM sleep, rhythmic slow waves are replaced by rapid, low-voltage irregular activity that elevates the threshold for arousal by sensory stimulation to the reticular activating system (RAS).[7]

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There are indications that REM sleep and dreaming may be dissociable states, and that, in this context, raises speculation about the similarities between dreaming and psychotic cognition.

The RAS is a polysynaptic-network, which perceives and analyzes divergent stimulus impulses and accordingly influences arousal and awareness mechanisms and sleep. Evolutionarily old, the RAS occupies the midventral portion of the medulla and midbrain and is an area made up of both specific and nonspecific fibers, interconnected with reciprocal acting inhibitory/ excitatory pathways.[7]

There are indications that REM sleep and dreaming may be dissociable states and, in this context, raises speculation about the similarities between dreaming and psychotic cognition.[11] Although it is not known how the impulses ascending in the RAS that disrupt synchronized cortical activity produce states of arousal, two centrally occurring mechanisms may explain the cause-effect link between REM sleep and perceptual disturbances.

The contribution of an over-reactive cortical cholinergic input system during REM sleep, with the possibility that increased dream-state impulse activity overrides to some degree RAS cortical inhibition may well lead to an increase in the stimulus-arousal quotient. These two interdependent schemes may be responsible for the reaction to emotionally charged dreams that lead to transitional arousal states and hypnogogic phenomena.

With regards,
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REFERENCES

1. Cheyne JA, Rueffer SD, Newby-Clark IR. Hypnogogic and hypnopompic hallucinations during sleep paralysis: Neurological and cultural construction of the nightmare. *Conscious Cogn* 1999;8(3): 319–37.
2. Schiller F. The inveterate paradox of dreaming. *Arch Neurol* 1985;42(9):903–6.
3. Neuropsychiatry, Neuropsychology, and Clinical Neuroscience. Emotion, Evolution, Cognition, Language, Memory, Brain Damage, and Abnormal Behavior. Second Edition 1996. Joseph, R. Section IV, The Brain Stem and The Basal Ganglia. P375.
4. Ohayon M, Priest R, Caulet M, Guilleminault C. Hypnogogic and hypnopompic hallucinations: Pathological phenomena? *Br J Psychiatry* 1996;169(4):459–67.
5. Ohayon M. Prevalence of hallucinations and their pathological associations in the general population. *Psychiatry Res* 2000; 27:97(2-3):153–64.
6. Hobson JA, Pace-Schott EF, Stickgold R. Dreaming and the brain: Toward a cognitive neuroscience of conscious states. *Behav Brain Sci* 2000;23(6):793–842.
7. William F. Ganong (ed). *Review of Medical Physiology, 17th Edition.* Norwalk, CT: Appleton & Lange, 1995:174–183
8. Rowland LP (ed). *Merritt's Textbook of Neurology, Eighth Edition*. Philadelphia, PA: Lea & Febiger, 1989:808–10.
9. Maquet P. Functional neuroimaging of normal human sleep by positron emission tomography. *J Sleep Res* 2000;9(3):207–31.
10. Sarter M, Bruno JP. Cortical cholinergic inputs mediating arousal, attentional processing and dreaming: differential afferent regulation of the basal forebrain by telencephalic and brainstem afferents. *Neuroscience* 2000;95(4):933–52.
11. Solms M. Dreaming and REM sleep are controlled by different brain mechanisms. *Behav Brain Sci* 2000; 23(6):843–50.